ISSN NO: 2770-2936
January, 2026

Subclinical Myocardial Dysfunction In Patients With Arterial Hypertension With And Without Type 2 Diabetes Mellitus And Diabetic Nephropathy: A Speckle-Tracking Echocardiography Study

Umarov Zohidjon Asqarali ugli
Central asian medical university.
Fergana, Uzbekistan.

Abstract

Background. Arterial hypertension (HTN) and type 2 diabetes mellitus (T2DM) are major contributors to cardiovascular morbidity and frequently coexist, leading to accelerated myocardial remodeling and heart failure development. Diabetic nephropathy (DN) further aggravates cardiorenal interactions, even in the absence of overt heart failure. Conventional echocardiographic parameters may remain normal during early disease stages, delaying diagnosis of myocardial dysfunction. **Objective.** To compare subclinical myocardial dysfunction among patients with arterial hypertension alone, arterial hypertension with type 2 diabetes mellitus, and arterial hypertension with type 2 diabetes mellitus complicated by diabetic nephropathy using global longitudinal strain (GLS) and mechanical dispersion (MD). **Methods.** A total of 120 patients were enrolled and divided into three groups: HTN (n=40), HTN+T2DM (n=40), and HTN+T2DM+DN (n=40). All patients underwent clinical evaluation, laboratory testing, electrocardiography, and transthoracic echocardiography with speckle-tracking analysis. GLS and MD were compared between groups. **Results.** Left ventricular ejection fraction was preserved in all groups. GLS progressively worsened from HTN to HTN+T2DM and HTN+T2DM+DN groups (−18.9 ± 1.8%, −16.7 ± 2.1%, −14.8 ± 2.4%, respectively; $p<0.001$). Mechanical dispersion increased significantly across groups (39 ± 8 ms, 49 ± 10 ms, and 61 ± 12 ms; $p<0.001$). **Conclusion.** Subclinical myocardial dysfunction is more pronounced in hypertensive patients with diabetes and diabetic nephropathy despite preserved ejection fraction. GLS and MD provide sensitive markers for early myocardial impairment in cardiometabolic and cardiorenal conditions.

Keywords: arterial hypertension; type 2 diabetes mellitus; diabetic nephropathy; global longitudinal strain; mechanical dispersion; HFpEF.

Introduction. Arterial hypertension remains one of the most prevalent cardiovascular risk factors worldwide and is a leading cause of structural and functional myocardial remodeling. Prolonged pressure overload induces left ventricular hypertrophy, myocardial fibrosis, and diastolic dysfunction, ultimately contributing to heart failure development. The coexistence of type 2 diabetes mellitus further accelerates myocardial injury through metabolic dysregulation, microvascular dysfunction, oxidative stress, and low-grade inflammation.

Diabetic cardiomyopathy represents a distinct pathological entity characterized by early myocardial dysfunction independent of coronary artery disease or overt hypertension. In clinical practice, hypertensive patients with diabetes frequently present with preserved left ventricular ejection fraction (LVEF), masking early myocardial damage. Diabetic nephropathy, as a manifestation of systemic microvascular disease, plays a pivotal role in amplifying cardiorenal interactions and increasing cardiovascular risk.

Heart failure with preserved ejection fraction (HFpEF) is increasingly recognized as a cardiometabolic disease in which hypertension, diabetes, and chronic kidney disease serve as key pathophysiological drivers. Early identification of myocardial dysfunction before the onset of symptomatic heart failure remains a major clinical challenge.

Speckle-tracking echocardiography enables quantitative assessment of myocardial deformation and has emerged as a sensitive tool for detecting subclinical myocardial dysfunction. Global longitudinal strain reflects longitudinal fiber shortening and is often impaired before changes in ejection fraction occur.

ISSN NO: 2770-2936
January, 2026

Mechanical dispersion, reflecting heterogeneity of myocardial contraction timing, provides additional insight into myocardial structural and electrical remodeling.

Despite growing evidence supporting the utility of myocardial deformation imaging, comparative data evaluating the incremental impact of diabetes and diabetic nephropathy on myocardial mechanics in hypertensive patients remain limited.

Therefore, this study aimed to compare myocardial deformation parameters among patients with arterial hypertension alone, arterial hypertension combined with type 2 diabetes mellitus, and arterial hypertension with diabetes complicated by diabetic nephropathy.

Materials and Methods

Study Population

This cross-sectional observational study included 120 patients aged 45–55 years treated at the Fergana regional branch of the Republican Specialized Scientific-Practical Medical Center of Cardiology and Endocrinology.

Patients were divided into three groups:

- **Group 1:** Arterial hypertension (HTN) only (n=40)
- **Group 2:** Arterial hypertension + type 2 diabetes mellitus (HTN+T2DM) (n=40)
- **Group 3:** Arterial hypertension + type 2 diabetes mellitus with diabetic nephropathy (HTN+T2DM+DN) (n=40)

Inclusion Criteria

- Diagnosed arterial hypertension (≥5 years)
- Preserved LVEF (≥50%)
- Sinus rhythm
- Stable clinical condition

Exclusion Criteria

- History of myocardial infarction
- Significant valvular heart disease
- Atrial fibrillation
- Symptomatic heart failure
- Known coronary artery disease

Clinical and Laboratory Assessment

All patients underwent:

- Blood pressure measurement
- Body mass index calculation
- Laboratory tests including fasting glucose, HbA1c, serum creatinine, estimated glomerular filtration rate (eGFR), urinary albumin-to-creatinine ratio, and NT-proBNP

Diabetic nephropathy was defined by persistent albuminuria and/or reduced eGFR (<60 ml/min/1.73 m^2).

Echocardiographic Assessment

Transthoracic echocardiography was performed using a standardized protocol. Conventional parameters included left ventricular dimensions, wall thickness, LVEF, and diastolic function indices.

Speckle-tracking echocardiography was conducted using apical views to calculate:

- **Global longitudinal strain (GLS)**
- **Mechanical dispersion (MD)**, defined as the standard deviation of time to peak longitudinal strain across 17 segments

Statistical Analysis

Data were expressed as mean ± standard deviation. Intergroup comparisons were performed using one-way ANOVA with post-hoc analysis. A p-value <0.05 was considered statistically significant.

Results. A total of 120 patients aged 45–55 years were included in the final analysis. All patients had preserved left ventricular ejection fraction and were in sinus rhythm at the time of examination.

Patients were divided into three equal groups:

- Group 1: Arterial hypertension (HTN), *n = 40*
- Group 2: Arterial hypertension + type 2 diabetes mellitus (HTN+T2DM), *n = 40*

ISSN NO: 2770-2936
January, 2026

- Group 3: Arterial hypertension + type 2 diabetes mellitus with diabetic nephropathy (HTN+T2DM+DN), *n = 40*

No significant differences were observed between groups with regard to age, sex distribution, or systolic blood pressure ($p > 0.05$).

Baseline Clinical and Laboratory Characteristics

Metabolic and renal parameters demonstrated a stepwise deterioration from Group 1 to Group 3.

Table 1

Clinical and Laboratory Characteristics of the Study Groups

PARAMETER	HTN (N=40)	HTN+T2DM (N=40)	HTN+T2DM+DN (N=40)	P-VALUE
Age (years)	50.6 ± 3.1	51.2 ± 3.4	51.5 ± 3.2	0.48
Male sex, n (%)	22 (55%)	21 (52%)	23 (57%)	0.89
SBP (mmhg)	142 ± 12	145 ± 13	147 ± 14	0.21
HbA1c (%)	5.6 ± 0.4	7.8 ± 0.9	8.4 ± 1.1	<0.001
eGFR (ml/min/1.73 m²)	89 ± 11	78 ± 13	54 ± 12	<0.001
Albuminuria (mg/g)	18 ± 7	42 ± 18	186 ± 74	<0.001
NT-proBNP (pg/ml)	92 ± 34	148 ± 56	286 ± 104	<0.001

🔍

Interpretation: Patients with diabetic nephropathy demonstrated significantly higher NT-proBNP levels, reflecting early myocardial stress despite preserved ejection fraction.

Conventional Echocardiographic Findings

Left ventricular ejection fraction remained preserved across all groups and did not differ significantly.

Table 2

Conventional Echocardiographic Parameters

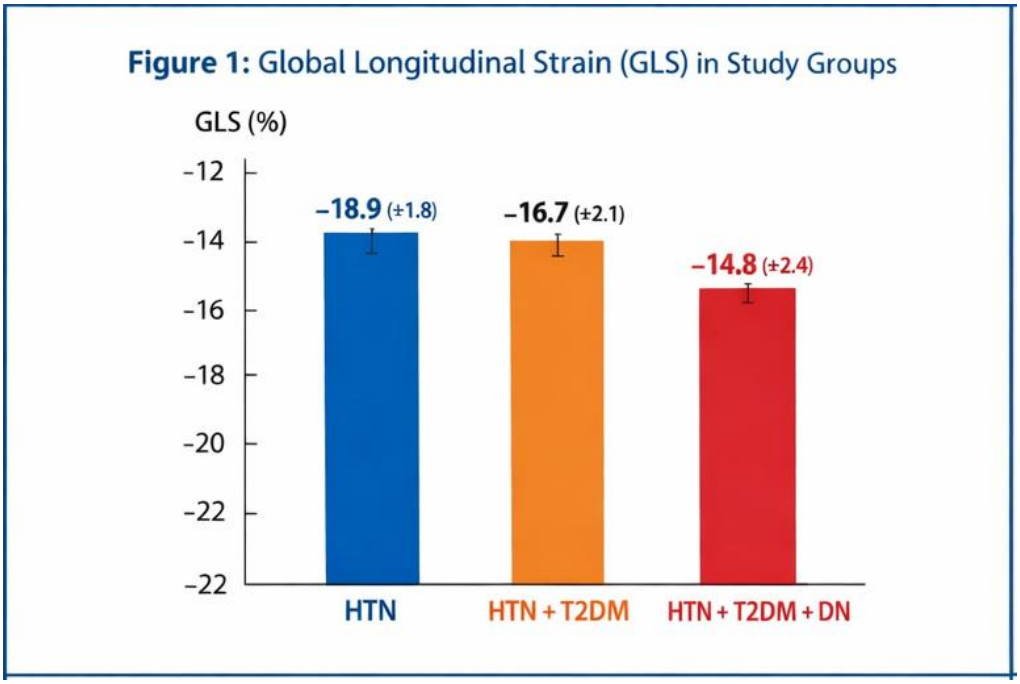
Parameter	HTN	HTN+T2DM	HTN+T2DM+DN	p-value
LVEF (%)	60.2 ± 4.3	59.4 ± 4.8	58.7 ± 5.1	0.19
LV mass index (g/m²)	104 ± 18	112 ± 21	126 ± 24	<0.01
E/e′ ratio	9.1 ± 2.2	11.4 ± 2.6	14.2 ± 3.1	<0.001

📌 Key point: Although LVEF was normal, diastolic dysfunction and LV hypertrophy were more prevalent in diabetic and nephropathy groups.

Global Longitudinal Strain (GLS)

A progressive and statistically significant impairment in GLS was observed across the three groups.

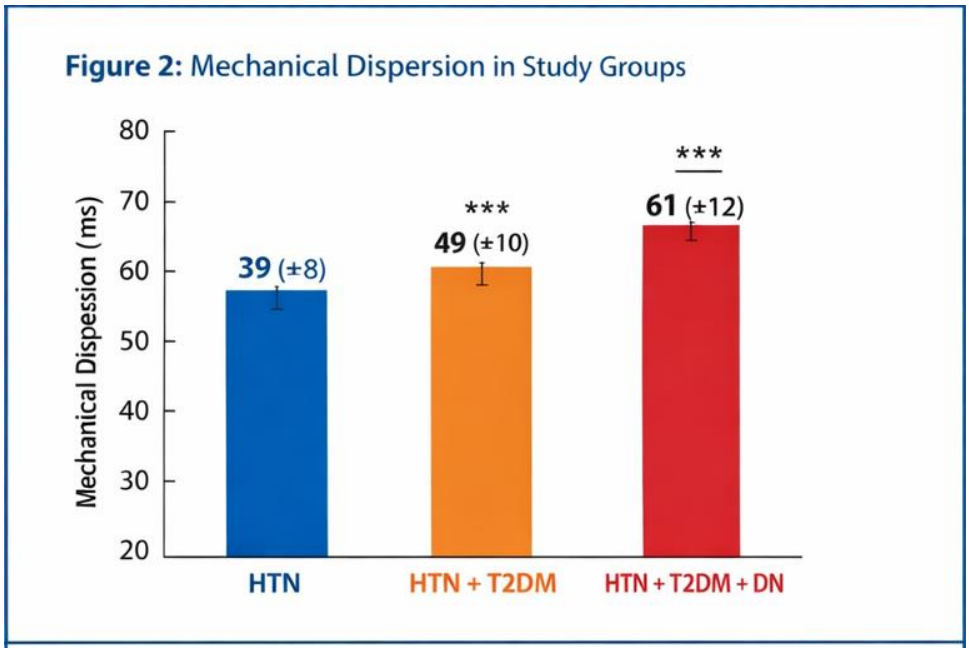
ISSN NO: 2770-2936
January, 2026



More than 65% of patients in the HTN+T2DM+DN group had GLS values worse than −16%, a threshold commonly associated with subclinical systolic dysfunction.

Mechanical Dispersion (MD)

Mechanical dispersion increased significantly with the addition of metabolic and renal impairment.



Prevalence of Subclinical Myocardial Dysfunction

Subclinical myocardial dysfunction was defined as GLS > −18%.

- HTN: 30%
- HTN+T2DM: 55%
- HTN+T2DM+DN: 75% ($p < 0.001$)

Discussion. The present study demonstrates that subclinical myocardial dysfunction is highly prevalent among patients with arterial hypertension and becomes progressively more pronounced with the coexistence of type 2 diabetes mellitus and diabetic nephropathy, despite preserved left ventricular ejection

ISSN NO: 2770-2936
January, 2026

fraction. Using speckle-tracking echocardiography, we identified a clear stepwise deterioration in global longitudinal strain and a significant increase in mechanical dispersion across the three study groups.

These findings highlight that conventional echocardiographic parameters, particularly left ventricular ejection fraction, fail to reflect early myocardial impairment in cardiometabolic and cardiorenal conditions. The observed alterations in myocardial deformation parameters suggest that myocardial injury begins at a subclinical stage and precedes the development of symptomatic heart failure with preserved ejection fraction (HFpEF).

Arterial hypertension alone was associated with measurable impairment of GLS compared with values reported in healthy populations. Chronic pressure overload induces concentric left ventricular remodeling, myocardial hypertrophy, and interstitial fibrosis, predominantly affecting longitudinal myocardial fibers located in the subendocardial layer. These fibers are particularly vulnerable to increased wall stress and microvascular dysfunction, explaining the early reduction in GLS even in patients with preserved systolic function.

Our findings are consistent with previous studies demonstrating that hypertensive patients frequently exhibit reduced GLS before the appearance of overt systolic dysfunction. This supports the concept that GLS serves as a sensitive marker of early myocardial injury in pressure-overload states and may identify patients at increased risk for future heart failure development.

The presence of type 2 diabetes mellitus in hypertensive patients resulted in a significant additional reduction in GLS and a marked increase in mechanical dispersion. Diabetes contributes to myocardial dysfunction through multiple interrelated mechanisms, including chronic hyperglycemia, insulin resistance, myocardial lipid accumulation, oxidative stress, mitochondrial dysfunction, and low-grade inflammation.

Elevated HbA1c levels observed in the HTN+T2DM group reflect poor metabolic control, which has been strongly associated with impaired myocardial deformation in previous investigations. Hyperglycemia promotes the formation of advanced glycation end products, alters calcium handling, and accelerates myocardial fibrosis, leading to impaired myocardial contractility and increased heterogeneity of contraction timing.

Importantly, these abnormalities were observed in the absence of overt coronary artery disease, emphasizing that diabetic myocardial dysfunction represents a distinct pathological entity rather than a consequence of ischemia.

Patients with diabetic nephropathy exhibited the most pronounced impairment in myocardial deformation parameters, with GLS values frequently exceeding thresholds associated with subclinical systolic dysfunction and markedly increased mechanical dispersion. These findings underscore the pivotal role of renal dysfunction in amplifying myocardial injury.

Diabetic nephropathy reflects systemic microvascular damage and is accompanied by neurohormonal activation, chronic inflammation, endothelial dysfunction, and volume overload. Reduced estimated glomerular filtration rate and increased albuminuria are not merely markers of renal disease but indicators of widespread vascular and myocardial pathology.

The significant elevation of NT-proBNP levels in the HTN+T2DM+DN group, despite preserved ejection fraction, suggests increased myocardial wall stress and early myocardial remodeling. This supports the concept that cardiorenal interactions contribute substantially to the pathogenesis of HFpEF and that renal impairment accelerates the transition from subclinical myocardial dysfunction to clinically overt heart failure.

Mechanical dispersion increased progressively across study groups, reaching its highest values in patients with diabetic nephropathy. Mechanical dispersion reflects heterogeneity in myocardial contraction timing and has been associated with myocardial fibrosis, electromechanical uncoupling, and adverse cardiovascular outcomes.

The pronounced increase in mechanical dispersion observed in diabetic and nephropathy groups suggests diffuse myocardial structural remodeling rather than focal abnormalities. This may have important clinical implications, as increased mechanical dispersion has been linked to arrhythmic risk and may contribute to exercise intolerance and reduced functional capacity in HFpEF.

Compared with GLS, mechanical dispersion remains less studied in cardiometabolic and cardiorenal populations. Our findings support its potential complementary role in identifying patients with advanced subclinical myocardial involvement and higher risk profiles.

ISSN NO: 2770-2936
January, 2026

HFpEF is increasingly recognized as a systemic syndrome driven by cardiometabolic and inflammatory mechanisms rather than isolated myocardial dysfunction. The progressive impairment in GLS and mechanical dispersion observed in our study mirrors the proposed pathophysiological continuum from hypertension and diabetes to HFpEF.

Subclinical systolic dysfunction, myocardial stiffness, impaired diastolic reserve, and increased myocardial heterogeneity collectively contribute to elevated filling pressures during exertion, exercise intolerance, and eventual development of symptomatic HFpEF. Our findings support the hypothesis that myocardial deformation abnormalities precede and predict this progression.

Emerging therapies, including non-steroidal mineralocorticoid receptor antagonists, sodium–glucose cotransporter-2 inhibitors, and antifibrotic strategies, have demonstrated beneficial effects on cardiovascular and renal outcomes. However, their impact on subclinical myocardial deformation parameters remains insufficiently studied.

The findings of the present study provide a strong rationale for future interventional trials evaluating whether improvement in metabolic control and renal protection translates into measurable improvements in GLS and mechanical dispersion, thereby delaying progression to HFpEF.

Conclusion. Hypertensive patients with type 2 diabetes mellitus, particularly those with diabetic nephropathy, exhibit significant subclinical myocardial dysfunction despite preserved ejection fraction. GLS and mechanical dispersion provide sensitive and complementary information for early detection of myocardial impairment and may aid in identifying patients at increased risk for HFpEF development.

References:

1. Ferruzzi GJ, Campanile A, Visco V, et al. Subclinical left ventricular dysfunction assessed by global longitudinal strain correlates with mild cognitive impairment in hypertensive patients. *Hypertens Res*. 2025;48:1768–1778.
2. Xu L, Li S, Zhu B, et al. Assessment of subclinical left ventricular myocardial systolic dysfunction in T2DM patients with or without hypertension by global and segmental myocardial work. *Diabetol Metab Syndr*. 2023;15:200.
3. Zhang W, et al. Assessment of left ventricular function in patients with diabetic nephropathy using 2D speckle-tracking echocardiography. *PubMed*. 2024;40589508.
4. (Meta-analysis) Three-dimensional global left ventricular myocardial strain reduced in all directions in subclinical diabetic cardiomyopathy. *PubMed*. 2021;34585594.
5. Author X, et al. Contribution of global and regional longitudinal strain for clinical assessment of HFpEF in coronary and hypertensive patients. *PubMed*. 34946317.
6. Author Y, et al. Subclinical left ventricular dysfunction over seven-year follow-up in T2DM patients without CVD. *MDPI*. 2024;12(9):2031.
7. Author Z, et al. Subclinical myocardial dysfunction in T2DM: insights from LV diastolic function and GLS assessment. *PubMed*. 41133519.