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Original article

GLYCEMIC CONTROL AND LEFT ATRIAL FUNCTION ASSESSED BY STRAIN-BASED ECHOCARDIOGRAPHY

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Introduction: Diabetes mellitus (DM) and prediabetes are associated with adverse cardiac remodeling. Left atrial (LA) strain, assessed by speckle-tracking echocardiography, can detect subtle LA dysfunction before structural changes occur.

Aim: To evaluate the relationship between glycemic control and LA strain in patients with type 2 DM and prediabetes.

Methods: We studied 155 consecutive patients with DM or prediabetes who underwent standard 2D echocardiography and LA strain analysis. Glycemic control was assessed by HbA1c and categorized as good or poor. LA reservoir (PALS) and contractile (PACS) strain were compared between groups. Correlation and multivariate regression analysis were performed to assess association between HbA1c and LA strain.

Results: Patients with poor glycemic control (n = 33) had significantly lower PALS (p=0.012) and higher prevalence of PALS values above the lowest normal (p=0.006) compared to those with good control. HbA1c was positively correlated with the presence of PALS and PACS above the lowest normal value. However, in multivariate regression, poor glycemic control did not retain independent predictive value for impaired LA strain after adjusting for age, BMI, LA volume, and/or LV global longitudinal strain (GLS%) in the whole cohort and in the prediabetic subgroup.

Conclusion: In patients with type 2 diabetes and prediabetes, poor glycemic control is associated with early LA dysfunction, as detected by strain analysis, even in the absence of LA enlargement or overt LV systolic impairment. LA strain may serve as an early marker of subclinical atrial remodeling in dysglycemia.

Keywords: diabetes mellitus, prediabetes, glycemic control, left atrial strain, speckle-tracking echocardiography

Introduction

Diabetes mellitus (DM) is a major global health problem, with a rising prevalence and high rates of disability and mortality[1]. Current estimates suggest that nearly half of individuals with DM (49.7%) remain undiagnosed[1]. In addition, people with prediabetes, defined by impaired glucose tolerance or impaired fasting glucose, are at high risk of developing DM, with up to 50% progressing within five years[2]. Together, these conditions form a continuum of metabolic risk with significant cardiovascular impact.

Chronic hyperglycemia, systemic insulin resistance, and impaired cardiac insulin signaling induce structural and functional changes in the myocardium and intracardiac structures. These alterations occur through multiple mechanisms, including myocardial fibrosis, inflammation, microvascular dysfunction, autonomic neuropathy, and other metabolic derangements[3-5]. Optimal glycemic control is therefore essential for the prevention and management of cardiovascular complications in patients with DM and prediabetes. Glycosylated hemoglobin (HbA1c) is the standard biomarker for assessing long-term glycemic control[5-7], but its relationship to early atrial mechanics in prediabetes and DM remains incompletely understood.

Echocardiographic assessment of left atrial (LA) function using strain imaging has recently emerged as a sensitive tool for detecting early atrial myocardial dysfunction, even before conventional echocardiographic parameters reveal abnormalities[8,9]. Given that the LA is particularly sensitive to metabolic and hemodynamic changes in DM, several studies have demonstrated impaired LA strain in patients with DM and prediabetes, linking poor glycemic control to atrial remodeling and dysfunction [10-13]. However, existing studies are limited by heterogeneous populations, varying definitions of glycemic control, and a predominant focus on established DM rather than the earlier prediabetic stage where prevention strategies may be most effective.

This study aimed to address these gaps by systematically evaluating the relationship between glycemic control, measured by HbA1c, and LA strain parameters obtained by speckle-tracking echocardiography in both prediabetic and diabetic patients. By integrating metabolic and imaging data, we aimed to determine whether LA strain can serve as an early, sensitive marker of subclinical atrial dysfunction across the spectrum of glucose dysregulation.

Material and methods

Study population

We conducted a cross-sectional study including 155 consecutive patients with type 2 DM (n=95) or prediabetes (n=60) referred for comprehensive echocardiographic evaluation between 2024 and 2025. DM was defined according to the American Diabetes Association (ADA) criteria: fasting plasma glucose ≥7mmol/L, and/or 2-hour plasma glucose ≥11.0 mmol/L during an oral glucose tolerance test, and/or HbA1c ≥6.5%[13]. Prediabetes was defined as fasting plasma glucose 5.6-6.9 mmol/L, and/or 2-hour plasma glucose 7.8-11.0 mmol/L during an oral glucose tolerance test, and/or HbA1c 5.7-6.4%[14].

Exclusion criteria were: age <30 years, known cardiovascular disease (except controlled hypertension), moderate or severe valvular heart disease, atrial fibrillation, estimated glomerular filtration rate (eGFR) <45 ml/min/1.73 m^2, pregnancy, significant renal or hepatic dysfunction, or inadequate echocardiographic image quality.

A written informed consent was provided by each participant of the study, and this study was approved by Institutional Review Boards at all participating institutions.

Clinical and laboratory assessment

Demographic and clinical variables, including age, sex, body surface area (BSA), body mass index (BMI), blood pressure, and heart rate were obtained at the time of echocardiographic examination. Venous blood samples were obtained after an overnight fast for determination of glucose, glycated hemoglobin (HbA1c), insulin level lipid profile, renal function, high-sensitivity C-reactive protein (hs-CRP, mg/L) and N-terminal pro-B-type natriuretic peptide (NT-proBNP, pg/mL).

Glycemic control was assessed using HbA1c both as a continuous variable and as a categorical variable: good control: HbA1c <7.0%, and poor control: HbA1c ≥7.0%.

All procedures were made in accordance with the Declaration of Helsinki.

Echocardiographic evaluation

All patients underwent a standard transthoracic echocardiography (system: GE-Vivid 7) according to the recommendations of the American Society of Echocardiography and the European Association of Cardiovascular Imaging[15-18]. Measured parameters included: LA size and left atrial volume index by BSA (LAVI), LA ejection fraction, LV ejection fraction (biplane Simpson method), LV mass indexed by BSA, LV global longitudinal strain (GLS%), E/e' ratio using Pulsed-waved and Tissue Doppler imaging (respectively), and LA function: peak atrial longitudinal strain (PALS) measured at the end of the reservoir phase and peak atrial contraction strain (PACS) or late diastolic strain, by two-dimensional speckle-tracking echocardiography from apical four- and two-chamber views using QRS as the reference point for the analysis.

LA strain analysis was performed using EchoPAC software (GE Medical System) at frame rates of 40-80 fps, with the average value from both views reported. All echocardiographic analyses were performed by two operators blinded to each other's findings, one of whom was a senior expert.

We adopted the normal reference values for LA strain from the Copenhagen City Heart Study, in which the median values and corresponding limits of normality for PALS and PACS were 39.4% (23.0-67.6) and 15.5% (6.4-28.0), respectively[18].

Reproducibility

To assess the reproducibility and reliability of the LA strain measurements, we calculated the intraclass correlation coefficient (ICC) for interobserver variability using 20 randomly selected images assessed in a blinded fashion on two separate occasions.

Statistical analysis

Continuous variables were expressed as mean ± standard deviation. Comparisons between groups (good *vs.* poor glycemic control) were performed using the Student's t-test or Mann-Whitney U test for continuous variables, and χ^2 test for categorical variables. Correlation analysis was performed using Pearson's method in order to assess the association between HbA1c and each LA parameter (LAV, LAVI, PALS, PACS). Variables with $p<0.05$ along with clinically relevant covariates, were entered into multivariable linear regression models to identify independent predictors of LA size and function. Subgroup analyses were performed separately for patients with prediabetes.

A two-tailed $p<0.05$ was considered statistically significant. Statistical analyses were performed using SPSS, version 25.0 (IBM SPSS, Inc. Chicago, IL, USA).

Results

The comparison of baseline characteristics between patients with DM and prediabetes, divided by HbA1c into poor control (n=33) and good control (n=122) groups, showed (Table 1) that poor glycemic control was significantly more frequent in patients with higher systolic BP ($p=0.014$), longer disease duration (107 *vs*. 37 months; $p<0.0001$), more inflammation (higher hsCRP, $p=0.045$), and less frequent physical activity (more "never" and fewer "occasional/ regular" exercisers) ($p=0.027$).

No significant differences were found in BMI, renal function, or NT-proBNP levels. All patients in the poor control group were on glucose-lowering therapy (*vs*. 72% in good control; $p<0.0001$), while cardiovascular medication use was similar between groups.

Table 1. Baseline characteristics by glycemic control group (poor vs. good)-demographics, risk factors, clinical assessment, medication, laboratory findings.

Parameters	Poor control n=33	Good control n=122	p
Age (years)	54.82 ± 8.60	52.93±9.07	0.287
Gender (n/%)	m 20/60.6, f 13/39.4	m 56/45.9, f 66/54.1	0.096
BMI kg/m2	30.89 ± 6,23	29.62 ± 4,94	0.219
Hypertension (n/%)	12/36,4	46/37,7	0.528
Smoking (n/%)	9/27.3	40/32.8	0.352
Physical activity			
Regular	12/36.4	26/21.3	
Occasional	8/24.2	61/50.0	0.027
Never	13/39.4	35/28.7	
BPs (mmHg)	129.39 ± 14.45	122.54 ± 13.96	0.014
BPd (mmHg)	79.09 ± 7.01	76.76 ± 8.37	0.146
HR (beats/min)	80.53 ± 15.55	75.61 ± 12.75	0.066
Prediabetes (n/%)	1/3	59/48.4	
Durat. of DM/pDM (months)	106.77 ± 115.20	36,93 ± 54.70	0.0001
Fasting plasma glucose (mmol/L)	10.58 ± 3.72	6.53 ± 1.44	0.0001
HbA1c%	8.50 ± 1.10	5.91 ± 0.48	0.0001
Insulin (mcU/ml)	31.22 ± 40.62	19.21 ± 30.96	0.068
Medication for hyperglic. (n/%)	33/100	88/72.1	0.0001
Medication for CVD[1] (n/%)	19/57.6	69/56.6	0.539
hsCRP (mg/L)	6.03 ± 11.76	3.52 ± 3.92	0.045
Nt-proBNP (pg/L)	77.91 ± 110.83	59.82 ± 56.21	0.207
eGFR (ml/min/1.73 m^2)	87.04 ± 15.66	90.56 ± 20.73	0.366

BMI=body mass index, BPs=blood pressure systolic, BP=blood pressure diastolic, HR=heart rate, DM=diabetes mellitus, pDM=prediabetes, HBA1c= glycated hemoglobin, CVD=cardiovascular disease, hsCRP=high sensitivity C-reactive protein, Nt-proBNP=N-terminal pro-B-type natriuretic peptide, eGFR=estimated glomerular filtration rate. [1] Medication for CVD=hypertension and/or dyslipidemia.

Echocardiographic findings

Echocardiographic findings (Table 2) showed no significant difference in LV ejection fraction (LVEF) or LV mass index between groups. LV global longitudinal strain (GLS%) was slightly worse in the poor control group, but this did not reach statistical significance (p = 0.072). The diastolic function marker E/e' was significantly higher in the poor control group (8.47 *vs*. 7.33; p = 0.002), indicating higher LV filling pressures.

Regarding LA size and function, patients with poor glycemic control had significantly larger LA volume (p = 0.027), while LA diameter, indexed LA volume, and LA ejection fraction were similar between groups. Strain analysis revealed that LA longitudinal strain (PALS%) was significantly lower in poor control group (p = 0.012), with a higher prevalence of abnormal PALS% values (below the lowest normal value; p = 0.006). Contractile strain (PACS%) did not differ significantly in mean values, but abnormal PACS% prevalence was higher in the poor control group (p = 0.045).

The ICC for global PALS average was 0.975 (95% CI:0.938-0.990) and for global PACS average was 0.971 (95% CI: 0.917–0.989).

Table 2. Echocardiographic measures in patients devided by glycemic control (poor vs. good)

Parameters	Poor control n=33	Good control n=122	p
LA (mm)	36.21 ± 2.75	35.79 ± 2.94	0.457
LAV (ml)	61.00 ± 18.58	54.26 ± 14.37	0.027
LAVi (ml/m2)	29.32 ± 7.77	27.54 ± 6.59	0.189
LAEF%	59.33 ± 7.20	60.23 ± 7.86	0.550
PALS%	27.04 ± 8.39	30.61 ± 6.80	0.012
PALS above LNV (n/%)	12/36.4	17/14.2	0.006
PACS%	15.48 ± 5.06	16.28 ± 3.66	0.310
PACS above LNV (n/%)	2/6.1	0	0.045
LVEF%	64.15 ± 4.69	65.32 ± 4.19	0.169
LVMi	88.07 ± 15.55	84.84 ± 15.21	0.283
LV GLS%	19.69 ± 2.53	20.79 ± 3.02	0.072
E/e’	8.47 ± 1.80	7.33 ± 1.61	0.002

LA=left atria, LAV=left atrial volume, LAVi- LA volume indexed by body surface area, LAEF=left atrial ejection fraction, PALS= peak atrial longitudinal strain, PACS= peak atrial contraction strain, LNV=lowest normal values, LVEF=left ventricular ejection fraction, LVMi=left ventricular mass indexed by BSA, LV GLS=left ventricular global longitudinal strain, E/e’= ratio of early mitral inflow velocity (E) measured by pulsed-wave Doppler with early diastolic mitral annular velocity (e’) measuered by tissue Doppler imaging.

Correlation and prediction

Correlation analysis (Table 3) showed that lower PALS% as a continuous variable was significantly related to older age, female gender, higher BMI, longer DM/prediabetes duration, antidiabetic medications use, higher blood pressure (systolic and diastolic), elevated HbA1c (both as a continuous variable and ≥ 7%) (Figure 1), larger LAV and LAVI, higher E/e’ ratio, and lower (less negative) GLS% (all $p < 0.05$). When we correlated PALS% above the normal lowest value (Table 3), the results were similar, except there was no significance in relation to female gender, LAVI, LAEF% and E/e’ ratio.

Table 3. Correlation of PALS% value with covariates and echocardiographic measurements that demonstrated significant correlation.

Parameters	PALS (%)	PALS (%) ALN
Age (years)	r=-0.398, p=0.0001	r=0.196, p=0.015
Gender (female)	r=0.200, p=0.013	-
BMI (kg/m^2)	r=-0.266, p=0.001	r=0.171, p=0.035
Durat. of DM/pDM (months)	r=-0.263, p=0.001	r=0.174, p=0.031
BPs (mmHg)	r=-0.294, p=0.001	r=0.205, p=0.011
BPd (mmHg)	r=-0.234, p=0.004	r=0.177, p=0.028
HbA1c (%)	r=-0.199, p=0.014	r=0.174, p=0.031
HBA1c ≥ 7%	r=-0.202, p=0.012	r=0.233, p=0.004
LAV (ml)	r=-0.363, p=0.0001	r=0.193, p=0.017
LAVI (ml/m^2)	r=-0.274, p=0.001	-
LAEF%	r=0.178 p=0.028	-
E/e’	r=-0.234, p=0.004	-
LV GLS%	r=0.328, p=0.0001	r=-0.292, p=0.0001

PALS= peak atrial longitudinal strain, ALN=above lowest normal value, BMI=body mass index, DM=diabetes mellitus, pDM=prediabetes, BPs=blood pressure systolic, BP=blood pressure diastolic, HBA1c= glycated hemoglobin, LAV=left atrial volume, LAVi- LA volume indexed by body surface area, LAEF=left atrial ejection fraction, E/e’= ratio of early mitral inflow velocity (E) measured by pulsed-wave Doppler with early diastolic mitral annular velocity (e’) measuered by tissue Doppler imaging, LV GLS=left ventricular global longitudinal strain.

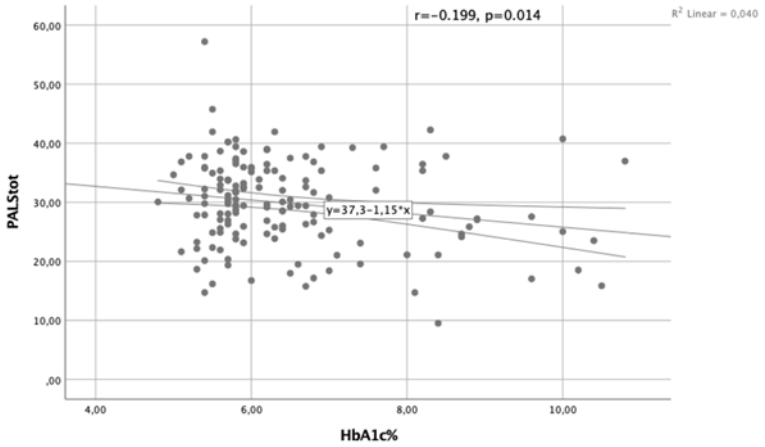

Fig. 1. Scatter plot of HbA1c *vs.* PALS
with fitted regression line (and adjusted R^2 annotation)

Table 4. Correlation of PACS% above normal lowest value with covariates and echocardiographic measurements that demonstrated significant correlation.

Parameters	PACS%	PACS (%) ALN
BMI (kg/m^2)	-	r=0.344, p=0.0001
Durat. of DM/pDM (months)	-	r=0.298, p=0.0001
HbA1c (%)	-	r=0.273, p=0.001
HBA1c ≥ 7%	-	r=0.219, p=0.006
Nt-proBNP (pg/L)	-	r=0.287, p=0.0001
LAV (ml)	r=-0.303, p=0.0001	-
LAVI (ml/m^2)	r=-0.251, p=0.002	-
LV GLS%	r=-0.248, p=0.003	r=-0.219, p=0.008

PACS= peak atrial contraction strain, ALN=above lowest normal value, BMI=body mass index, DM=diabetes mellitus, pDM=prediabetes, BPs=blood pressure systolic, BP=blood pressure diastolic, HBA1c= glycated hemoglobin, Nt-proBNP=N-terminal pro-B-type natriuretic peptide, LV GLS=left ventricular global longitudinal strain.

The correlation analysis of PACS% (Table 4) as a continuous variable demonstrated that its lower value was significantly related to dilated LAV and LAVI indexed and lower (less negative) GLS% (all $p<0.05$). When we correlated PACS% above the lowest normal value (Table 4), the analysis showed that its presence was associated with higher BMI, longer duration of DM or prediabetes, higher HbA1c (both as a continuous variable and ≥7%), higher level of Nt-proBNP and less negative (lower) GLS% (all $p < 0.05$).

All significant covariates were put into multivariable linear (for continuous variable) or logistic (for above the lowest normal value; binary) regression analysis. In the final step of the multivariable linear regression (Tables 5-6), the following independent predictors were identified:

PALS% (continuous) (Table 5): lower PALS was independently predicted by older age (B=-0.293, p=0.0001), lower GLS% (B=1.020, p=0.0001) and larger LAV (B=-0.144, p=0.0001).

In the prediabetes subgroup, GLS%, age, and LAV remained significant predictors ($p < 0.01$), with GLS% (B = 1.094, p=0.0001) being most predictive.

PACS% (continuous) (Table 5): lower PACS was predicted by larger LAV (B=-0.086, p= 0.0001) and less negative (lower) GLS% (B= 0.391, p=0.0001). In prediabetes subgroup, LAV and GLS remained significant predictors ($p < 0.05$).

Table 5. Multivariable linear regression analysis at last step for predictors of left atrial longitudinal strain (PALS), left atrial contraction strain (PACS) as a continuous variable.

Variable	B (unstandardized)	95%CI for B	Beta (Standardized)	Sig.
PALS%				
Age (years)	-0.293	-0.404 to -0.181	-0.354	0.0001
GLS%	1.020	0.708 to 1.332	0.421	0.0001
LAV (ml)	-0.144	-0.206 to -0.083	-0.318	0.0001
PALS% in prediabetes				
GLS%	1.094	0.636 to 1.551	0.479	0.0001
Age (years)	-0.302	-0.464 to -0.141	-0.392	0.0001
LAV (ml)	-0.148	-0.254 to -0.043	-0.291	0.007
PACS%				
LAV (ml)	-0.086	-0.123 to -0.048	-0.345	0.0001
GLS%	0.391	0.191 to 0.590	0.294	0.0001
PACS% in prediabetes				
LAV (ml)	-0.094	-0.160 to -0.028	-0.346	0.006
GLS%	0.359	0.065 to 0.654	0.294	0.018

GLS%=left ventricular global longitudinal strain, LAV=left atrial volume.

Table 6. Multivariable logistic regression analysis at last step for predictors of left atrial longitudinal strain (PALS) and left atrial contraction strain (PACS) above lowest normal values (ALN).

Variable	B	95%CI for B	Exp (B)	Sig.
PALS% LNV				
Age (years)	0.082	1.012 to 1.164	1.086	0.021
LAV (ml)	0.039	1.007 to 1.075	1.040	0.019
GLS%	-0.456	0.506 to 0.795	0.634	0.0001
PALS% LNV in prediabetes				
Age (years)	0.146	1.003 to 1.336	1.157	0.046
GLS%	-0.833	0.220 to 0.860	0.435	0.017
PACS%				
BMI (kg/m2)	2.392	0 to 6.433E+175	10.934	0.991

LAV=left atrial volume, GLS=left ventricular global longitudinal strain, BMI=body mass index.

PALS% lowest normal value (binary) (Table 6): abnormal PALS was predicted by older age (OR=1,086, p=0.021), larger LAV (OR=1.040, p=0.019) and less negative (lower) GLS% (OR=0.634, p=0.0001).

In prediabetes subgroup, LAV has lost its predictivity, thus older age increased the odds of having abnormal PALS% (OR=1.157, p=0.046), while less negative (lower) GLS% remained as an independent predictor (OR=1.435, p=0.017).

PACS% lowest normal value (binary) (Table 6): only BMI was retained as a predictor, but with wide confidence intervals and borderline significance.

Discussion

In this cross-sectional study including patients with type 2 DM and prediabetes, we found that poorer glycemic control, reflected by higher HbA1c levels, was associated with impaired left atrial (LA) reservoir function (PALS) and, to a lesser extent, contractile function (PACS), independent of conventional echocardiographic parameters. However, poor glycemic control did not retain its independent predictive value for impaired LA strain in the final steps of regression analysis, where age, BMI, LA volume (ml) and/or LV global longitudinal strain (GLS%) emerged as stronger predictors in both the overall cohort and the prediabetic subgroup. Importantly, LA strain impairment was detectable in the absence of overt LV systolic dysfunction, suggesting that LA strain may serve as an early marker of diabetic atrial myopathy across the spectrum of glucose dysregulation.

Our findings align with previous reports indicating that DM is associated with adverse atrial remodeling and dysfunction. Mondillo *et al.*[9] demonstrated that hypertensive and diabetic patients can exhibit early LA strain abnormalities despite preserved LA size and LV systolic function. Georgievska-Ismail *et al.*[10] similarly found that global PALS and PACS were significantly reduced in DM patients compared to non-diabetic controls, supporting the concept that DM-related atrial myiopathy contributes to functional decline. Muranaka *et al.* [19] also suggested that fibrotic changes in the LA in DM are responsible for reduced phasic function, as measured by strain rate parameters. Tadic and Cuspidi[11] also observed structural and functional LA changes in type 2 diabetes, attributable to both metabolic and hemodynamic factors. More recently, Garg *et al*.[12], in the ARIC cohort, reported that higher HbA1c levels were independently associated with impaired LA function, even after adjustment for cardiovascular risk factors and LV diastolic function. However, most prior studies focused exclusively on established DM. Our study extends these observations to prediabetes, a stage in which up to 50% of individuals progress to overt diabetes within five years[2], highlighting the importance of early detection of subclinical cardiovascular damage.

The mechanisms linking poor glycemic control to LA dysfunction are multifactorial. Chronic hyperglycemia, insulin resistance, and impaired myocardial insulin signaling promote interstitial fibrosis, low-grade inflammation, microvascular rarefaction, oxidative stress, and autonomic imbalance[3-5]. These processes increase LA stiffness, reduce compliance, and impair reservoir and contractile function. The LA may be particularly vulnerable due to its thin wall, high collagen content, and continuous exposure to LV filling pressures. In our cohort, poor glycemic control was associated with a higher E/e′ ratio and less negative GLS% values, suggesting that both diastolic dysfunction and subtle LV systolic impairment may contribute to LA strain abnormalities. This is consistent with the findings of Antit *et al*.[19], who showed that LA strain predicts elevated LV filling pressure in patients with preserved systolic function, with good sensitivity and specificity. Similar pathophysiological links between metabolic dysregulation, diastolic dysfunction, and LA mechanics have been emphasized in recent reviews[8,10,16]. Moreover, Gao *et al*.[21] demonstrated in 292 patients with type 2 DM that LV GLS% was independently related with HbA1c level and was not influenced by LA function.
From a clinical standpoint, our results underscore the potential role of LA strain as a sensitive imaging biomarker for early atrial dysfunction in patients with abnormal glucose metabolism. Detecting LA strain impairment in prediabetes or in diabetics with good LV systolic function could prompt more aggressive lifestyle or pharmacologic interventions to optimize glycemic control and potentially prevent structural remodeling. Given that LA dysfunction is a predictor of atrial fibrillation, stroke, and heart failure[8,16] , integration LA strain assessment into routine echocardiographic evaluation for high-risk metabolic patients could enhance cardiovascular risk stratification. In this respect, Tolvaj *et al.*[22] recently suggested incorporating PALS into the first-line echocardiographic assessment of diastolic function, given its ability to improved classification and risk stratification.

Strengths of our study include the simultaneous evaluation of LA reservoir and contractile function, blinded and reproducible strain measurements, and inclusion of both diabetes and prediabetes. However, several limitations should be acknowledged. First, the cross-sectional design precludes causal inference. Second, echocardiographic data were obtained from a single center. Third, we did not use dedicated software for LA strain analysis. Finally, the lack of follow-up limits our ability to determine whether LA strain impairment predicts future clinical events. Longitudinal studies are needed to assess whether improving glycemic control leads to measurable improvement or stabilization of LA strain parameters.

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Conclusion

Impaired LA reservoir function is closely associated with poor glycemic control in both diabetes and prediabetes, independent of LV systolic function. LA strain imaging may provide incremental value in detecting subclinical atrial dysfunction early in the course of glucose dysregulation, offering an opportunity for timely intervention to prevent progression to overt cardiovascular disease.

Conflict of interest statement. None declared.

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