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Divergent Effects of Glycemic Control in Diabetic Heart Failure: Arterial Stiffness vs. Subclinical Atrial Fibrillation

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ABSTRACT

Background: Diabetes mellitus and heart failure frequently coexist and contribute to vascular remodeling and arrhythmogenesis. While poor glycemic control promotes arterial stiffening, its relationship with device-detected subclinical atrial fibrillation (SCAF) remains unclear, particularly in longitudinal settings.

Objectives: To evaluate the impact of glycemic control on arterial stiffness and SCAF in patients with diabetes and heart failure with implanted cardiac devices.

Methods: In this prospective observational study, 86 patients with heart failure and type 2 diabetes were stratified by baseline HbA1c into strict control (<7.0%, n=29) and inadequate control (≥7.0%, n=57). Carotid–femoral pulse wave velocity (cf-PWV) and device-detected arrhythmias were assessed at baseline and 6 months. Group comparisons and regression analyses were performed using standard statistical methods.

Results: Baseline cf-PWV was higher in the inadequate control group [12.0 (9.7–15.0) vs. 9.4 (8.6–11.8) m/s; p=0.009], remaining higher at follow-up (p=0.002). cf-PWV correlated with HbA1c (r=0.250, p=0.01) and diabetes duration (r=0.296, p=0.006). Change in PWV did not differ between groups (p>0.05). SCAF incidence was greater in the strict control group (27.6% vs. 8.8%; p=0.029). Patients with SCAF had lower baseline HbA1c (6.9±0.7% vs. 7.9±1.5%; p=0.004). No difference was observed in ventricular arrhythmias or ICD therapies (p>0.05).

Conclusion: Poor glycemic control is associated with increased arterial stiffness, whereas strict control is linked to higher SCAF incidence in diabetic heart failure. These findings support individualized glycemic targets balancing vascular protection and arrhythmic risk.

Introduction

Diabetes mellitus (DM) and heart failure (HF) frequently coexist and act synergistically to accelerate cardiovascular remodeling, vascular dysfunction, and arrhythmogenesis [1]. Patients with HF and DM have a significantly higher risk of adverse cardiovascular outcomes, including hospitalization, stroke, and mortality, compared with those without diabetes [2-3] . Despite advances in pharmacological therapy and device-based management, residual cardiovascular risk in this population remains substantial, underscoring the need to identify modifiable pathophysiological drivers and early markers of disease progression.

Poor glycemic control is a central contributor to vascular injury through mechanisms involving endothelial dysfunction, oxidative stress, chronic low-grade inflammation, and advanced glycation end-product accumulation.[4] These processes promote arterial stiffening, which can be reliably quantified using carotid–femoral pulse wave velocity (cf-PWV), the gold standard measure of central arterial stiffness.[5-6] Increased arterial stiffness not only worsens ventricular–vascular coupling and left ventricular afterload but also leads to left atrial remodeling, thereby creating a substrate for atrial arrhythmias.

Subclinical atrial fibrillation (SCAF), detected by implantable cardiac devices as atrial high-rate episodes, is increasingly recognized as a clinically meaningful arrhythmic phenotype associated with stroke, heart failure decompensation, and mortality [7]. Patients with HF and DM are particularly vulnerable to SCAF due to structural atrial remodeling, autonomic imbalance, and microvascular dysfunction. [8] However, the interaction between glycemic control, arterial stiffness, and device-detected SCAF remains poorly characterized, especially in longitudinal settings.

Importantly, glycemic control is a dynamic process that may improve or deteriorate over time, yet most previous studies have relied on single baseline HbA1c measurements [9-10]. Whether changes in glycemic status translate into parallel changes in arterial stiffness and arrhythmic burden in patients with HF and DM is currently unknown.

Therefore, the present prospective observational study aimed to investigate the impact of glycemic control on arterial stiffness and device-detected subclinical atrial fibrillation in patients with diabetes

and heart failure, with a particular focus on the longitudinal changes in HbA1c and their relationship with vascular and arrhythmic outcomes.

Methods

Study Design and Patient Population

This prospective, single-center observational study included 86 consecutive patients with heart failure (HF) and diabetes mellitus who had an implanted an implantable cardioverter-defibrillator (ICD) or a cardiac resynchronization therapy defibrillator (CRT-D) device equipped with an atrial lead. The study protocol adhered strictly to the ethical principles outlined in the Declaration of Helsinki and received approval from the Institutional Review Board of Eskişehir Osmangazi University Faculty of Medicine (Decision No: 30; Date: November 3, 2020). Written informed consent was obtained from all participants prior to enrollment.

Inclusion criteria comprised patients aged ≥18 years with stable heart failure and a functional atrial lead capable of continuous rhythm monitoring. Exclusion criteria were defined as: (i) presence of permanent atrial fibrillation (AF); (ii) inability to tolerate the supine position for arterial stiffness assessment; (iii) pregnancy; (iv) New York Heart Association (NYHA) Class IV heart failure; and (v) incomplete data regarding echocardiography, carotid–femoral pulse wave velocity (cf-PWV), or device interrogation.

At baseline, the cohort was stratified into two groups based on glycemic control status: the "Strict Control" group (HbA1c <7.0%; n=29) and the "Inadequate Control" group (HbA1c ≥7.0%; n=57). Follow-up evaluations were conducted at 6 months. During this period, 11 patients were excluded from the final analysis due to death (n=4) or loss to follow-up (n=7). Consequently, 75 patients underwent the complete reassessment protocol, including repeat echocardiography, hemodynamic profiling, and device interrogation (Figure 1).

Assessment of Arterial Stiffness

Arterial stiffness was assessed non-invasively via carotid–femoral pulse wave velocity (cf-PWV), recognized as the gold-standard measure of aortic stiffness [5-6]. Measurements were performed using the SphygmoCor system (AtCor Medical, Sydney, Australia) employing high-fidelity applanation tonometry. Sequential pulse waveforms were recorded from the carotid and femoral arteries, gated to the R-wave of the simultaneous electrocardiogram (ECG).

The transit time between the two arterial sites was determined using the "foot-to-foot" method and the intersecting tangent algorithm. The path length was estimated by subtracting the distance between the suprasternal notch and the carotid sampling site from the distance between the suprasternal notch and the femoral sampling site, in accordance with standard consensus guidelines [11]. To evaluate the progression of vascular stiffness, the change in pulse wave velocity Δcf-PWV was calculated as the difference between the 6-month follow-up value and the baseline value.

Device Interrogation and Arrhythmia Analysis

Cardiac Implantable Electronic Devices (CIEDs) from major manufacturers (Medtronic, Abbott, Biotronik, and Boston Scientific) were interrogated using their respective proprietary programmers. Arrhythmic

burden was evaluated retrospectively for the 6 months preceding enrollment (baseline) and prospectively for the 6-month follow-up period.

The primary arrhythmic endpoint was subclinical atrial fibrillation (SCAF), defined as device-detected episodes of rapid atrial rate (≥190 beats per minute) persisting for >6 minutes, irrespective of symptoms. [12] Additionally, the burden of ventricular arrhythmias was quantified, including non-sustained and sustained ventricular tachycardia (VT), as well as device therapies (anti-tachycardia pacing [ATP] or shock delivery). To ensure diagnostic accuracy, only intracardiac electrograms with adequate atrial sensing and stable lead impedance were included in the analysis.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 21.0 (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for normality using the Shapiro-Wilk test and visual inspection of histograms. Normally distributed data are presented as mean ± standard deviation (SD) and were compared using the Student's t-test. Non-normally distributed variables are expressed as median with interquartile range (IQR) and were compared using the Mann–Whitney U test. Categorical data are summarized as frequencies and percentages, with group comparisons performed using the Chi-square test or Fisher's exact test, as appropriate.

Longitudinal changes within groups (Baseline vs. 6-Month) were analyzed using the paired-samples t-test for parametric data and the Wilcoxon signed-rank test for non-parametric data. Univariate linear regression analysis was conducted to identify potential predictors of arterial stiffness progression (Δcf-PWV) and arrhythmic events. Variables demonstrating a trend toward significance ($p<0.10$) in univariate analysis were entered into a multivariate linear regression model to determine independent predictors. Logistic regression analysis was performed to identify predictors of incident subclinical atrial fibrillation (SCAF), given its binary outcome structure.

All statistical tests were two-tailed, and a p-value of <0.05 was considered statistically significant.

Results

Baseline Clinical and Demographic Characteristics

The study cohort comprised 86 patients, stratified into two groups based on glycemic control: the strict control group (HbA1c <7.0%; n=29, 33.7%) and the inadequate control group (HbA1c ≥7.0%; n=57, 66.3%). Baseline demographic and clinical characteristics are summarized in Table 1. The groups were well-balanced regarding age, sex distribution, body mass index (BMI), and left ventricular ejection fraction (LVEF), with no statistically significant differences observed ($p>0.05$). However, as expected by the stratification criteria, the inadequate control group exhibited a significantly more adverse metabolic profile, characterized by higher fasting glucose and HbA1c levels, alongside lower HDL cholesterol levels ($p<0.05$). Regarding pharmacological management, the use of insulin and statin therapy was significantly more frequent in the high HbA1c group ($p<0.001$ and $p=0.037$, respectively).

Arterial Stiffness Assessment

Baseline aortic stiffness, assessed via cf-PWV, was significantly elevated in the inadequate control group compared to the strict control group [Median: 12.0 (9.7–15.0) m/s vs. 9.4 (8.6–11.8) m/s; p=0.009] (Figure 2). This disparity persisted at the 6-month follow-up, where cf-PWV values remained significantly higher in patients with HbA1c ≥7.0% (p=0.002). Correlation analysis revealed that baseline cf-PWV was positively correlated with both HbA1c levels (r=0.250, p=0.01) and diabetes duration (r=0.296, p=0.006) (Figure 3 and Figure 4). Longitudinally, however, the absolute change in arterial stiffness (Δcf-PWV) over the 6-month period did not differ significantly between the two groups (p>0.05), and no correlation was observed between changes in HbA1c (ΔHbA1c) and changes in PWV (ΔPWV) (r=–0.049, p=0.676).

Device-Detected Arrhythmic Events

At the 6-month device interrogation, a notable inverse association was observed between glycemic control and atrial arrhythmias. The incidence of new-onset subclinical atrial fibrillation (SCAF) was significantly higher in the strict control group (HbA1c <7.0%) compared to the inadequate control group (27.6% vs. 8.8%; p=0.029). Conversely, the incidence of ventricular tachycardia and appropriate ICD therapies (ATP or shock) showed no significant difference between the groups (Table 2).

When the cohort was re-stratified based on the presence of SCAF, patients who developed SCAF were found to have significantly lower baseline HbA1c (6.9 ± 0.7% vs. 7.9 ± 1.5%; p=0.004) and lower fasting glucose levels (131 [94–160] mg/dL vs. 161 [132–217] mg/dL; p=0.031) compared to those without SCAF (Table 3). Baseline cf-PWV did not differ significantly between patients with and without subclinical AF (12.7 ± 7.2 m/s vs. 12.1 ± 4.5 m/s; p=0.789).

Predictors of Arterial Stiffness Progression

In the high-risk subgroup (HbA1c ≥7.0%), univariate analysis identified baseline AF, left atrial (LA) volume and baseline cf-PWV as significant predictors of arterial stiffness progression (Δcf-PWV). In the multivariate linear regression model, baseline AF (β=+6.21, p=0.009), LA volume (β=+0.06, p=0.024), and baseline cf-PWV (β=–0.37, p=0.029) remained independent determinants of Δcf-PWV. Interestingly, traditional risk factors such as age and diabetes duration were not significantly associated with PWV progression in the multivariate model (Table 4).

Discussion

In this prospective observational study involving a high-risk cohort of patients with heart failure (HF) and type 2 diabetes mellitus (T2DM), we investigated the complex interplay between glycemic control, arterial stiffness, and device-detected subclinical arrhythmias. Our results revealed a striking dichotomy: while poor glycemic control (HbA1c ≥7.0%) was strongly associated with increased aortic stiffness, strict glycemic control (HbA1c <7.0%) was unexpectedly linked to a significantly higher incidence of subclinical atrial fibrillation (SCAF). This divergence challenges the conventional "lower is better" paradigm in diabetic heart failure, suggesting that the optimal therapeutic window may be narrower than previously assumed.

Our finding that patients with inadequate glycemic control exhibit significantly higher carotid-femoral pulse wave velocity (cf-PWV) reinforces the concept that chronic hyperglycemia functions as a potent accelerator of vascular aging. This observation aligns with the foundational evidence from the UK Prospective Diabetes Study (UKPDS), which established the causal link between hyperglycemia and microvascular deterioration [13]. The underlying pathophysiology likely involves the non-enzymatic glycation of proteins within the arterial wall, leading to the accumulation of advanced glycation end-products. [14] These byproducts promote collagen cross-linking and impair endothelial nitric oxide bioavailability, resulting in irreversible structural stiffening [15]. The strong positive correlation we observed between diabetes duration and cf-PWV further supports the "metabolic memory" hypothesis, indicating that arterial stiffness in this population is a reflection of cumulative long-term oxidative stress rather than transient glycemic fluctuations [16-17]. Consequently, preventing hyperglycemia remains crucial for mitigating vascular stiffening.

However, the most pivotal and novel finding of our study is the inverse association between HbA1c levels and device-detected SCAF. Contrary to traditional observational data linking diabetes to AF [18-19], our cohort demonstrated that patients with strict control (<7.0%) had a nearly three-fold higher incidence of SCAF compared to those with poorer control. This "glycemic paradox" strongly echoes the U-shaped relationship between glucose levels and mortality observed in major trials like ACCORD and ADVANCE [20-21-22]. In the specific context of heart failure, the myocardium is exceptionally vulnerable to metabolic instability. We hypothesize that the strict control group, despite having "ideal" HbA1c values, may have experienced greater glycemic variability or unrecognized hypoglycemic episodes. Severe hypoglycemia has also been associated with new-onset atrial fibrillation in population-based studies [23], and associations between antihyperglycemic therapies and AF risk have been reported in elderly patients] [24]. Hypoglycemia triggers a profound sympathoadrenal surge, resulting in catecholamine release and hypokalemia, which can prolong cardiac repolarization and precipitate atrial arrhythmias [25]. Given the high prevalence of autonomic neuropathy in diabetic HF patients, the counter-regulatory response to hypoglycemia is often blunted, potentially masking symptoms while leaving the electrical substrate exposed to arrhythmogenic triggers.

Furthermore, our longitudinal analysis provides critical insight into the bidirectional relationship between vascular and electrical remodeling. We identified that baseline AF and left atrial (LA) volume were independent predictors of the progression of arterial stiffness (ΔPWV)in strict control group . This suggests that SCAF is not merely a passive consequence of vascular disease but an active contributor to systemic vascular deterioration. The loss of atrial systole ("atrial kick") and the irregularity of the ventricular cycle length in AF may disrupt laminar blood flow, increasing shear stress on the arterial wall and accelerating stiffening [26-27] . Thus, a vicious cycle ensues: vascular stiffness increases cardiac afterload promoting atrial remodeling, while the resultant AF further impairs arterial compliance.

These findings have significant clinical implications, suggesting that the "one-size-fits-all" approach to glycemic targets—advocated by some guidelines—may be hazardous in patients with HF and implanted devices. While an HbA1c <7.0% is beneficial for microvascular protection [10], it may inadvertently increase the burden of subclinical arrhythmias if achieved at the cost of hypoglycemic excursions. Therefore, clinicians should consider a more personalized strategy, potentially favoring glucose-lowering

agents that minimize hypoglycemia and variability, such as SGLT2 inhibitors, over those that induce rapid glucose shifts.

Our study must be interpreted in light of certain limitations. The single-center design and sample size may limit generalizability. Additionally, while we utilized continuous device monitoring for precise arrhythmia detection—a major strength over Holter monitoring—we lacked continuous glucose monitoring (CGM) data to definitively correlate specific arrhythmic events with instantaneous glucose levels. Nevertheless, the use of gold-standard cf-PWV and the rigorous adjudication of device-detected arrhythmias provide robust evidence supporting our conclusions.

Conclusions

In conclusion, glycemic status showed divergent associations with vascular and arrhythmic outcomes in diabetic heart failure: higher HbA1c correlated with increased arterial stiffness, while lower HbA1c was linked to greater SCAF incidence. These results underscore the need for individualized metabolic targets in this high-risk population.

Declarations

Abbreviations

AF: Atrial fibrillation; ATP: Anti-tachycardia pacing; BMI: Body mass index; cf-PWV: Carotid–femoral pulse wave velocity; CIED: Cardiac implantable electronic device; CRT-D: Cardiac resynchronization therapy defibrillator; DM: Diabetes mellitus; HbA1c: Glycated hemoglobin; HF: Heart failure; ICD: Implantable cardioverter-defibrillator; LA: Left atrium; LVEF: Left ventricular ejection fraction; NYHA: New York Heart Association; SCAF: Subclinical atrial fibrillation; VT: Ventricular tachycardia.

Ethics approval and consent to participate

The study protocol was approved by the Institutional Review Board of Eskişehir Osmangazi University Faculty of Medicine (Decision No. 30; November 3, 2020). The study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrollment.

Consent for publication

Not applicable.

Availability of data and materials

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request, subject to applicable ethical and institutional restrictions.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

F.E.D., K.U.M., and G.Y. contributed to the study conception and design. K.U.M., G.Y., M.D., and S.M. supervised the study. F.E.D., O.O., E.B., and G.O.Y. contributed to resources. M.D., S.M., F.E.D., E.B., G.O.Y., and O.O. contributed to materials. F.E.D., O.O., E.B., G.O.Y., S.M., and G.Y. participated in data collection and processing. F.E.D. and K.U.M. performed data analysis and interpretation. All authors contributed to the literature review. F.E.D., O.O., E.B., S.M., G.Y., M.D., and K.U.M. contributed to manuscript writing. K.U.M., G.Y., M.D., S.M., and G.O.Y. critically reviewed the manuscript. All authors read and approved the final manuscript.

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Figure Legends:

Figure 1: Flow chart of study population

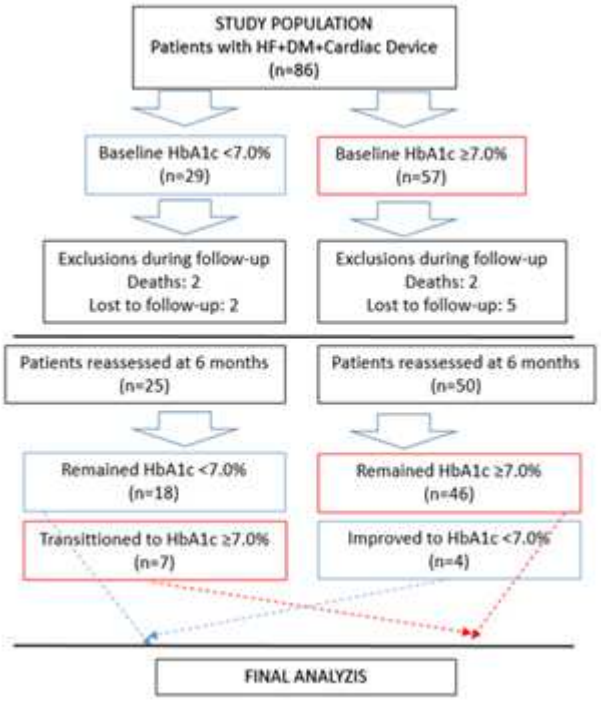


Figure 2: Baseline and 6-month cf-PWV values according to HbA1c groups

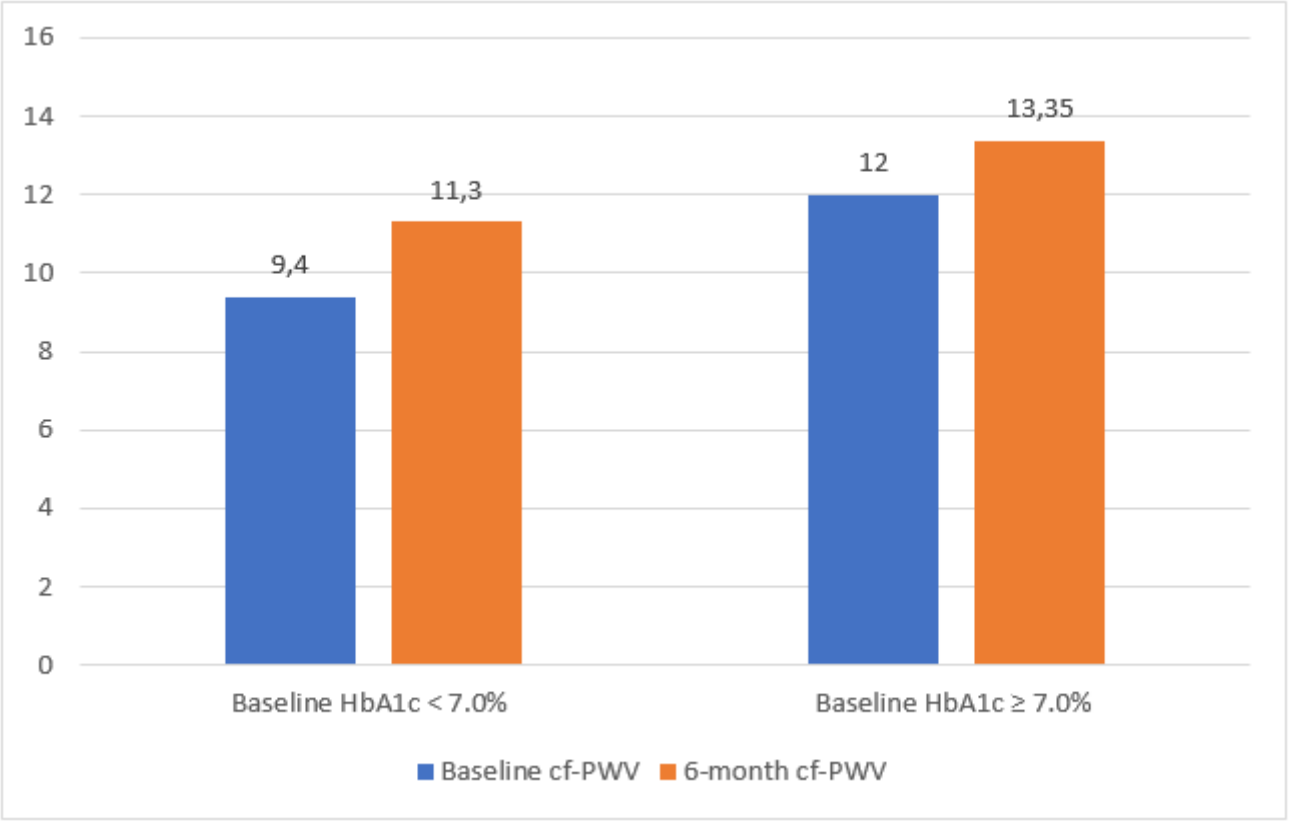


Figure 3: Correlation between HbA1c and cf-PWV

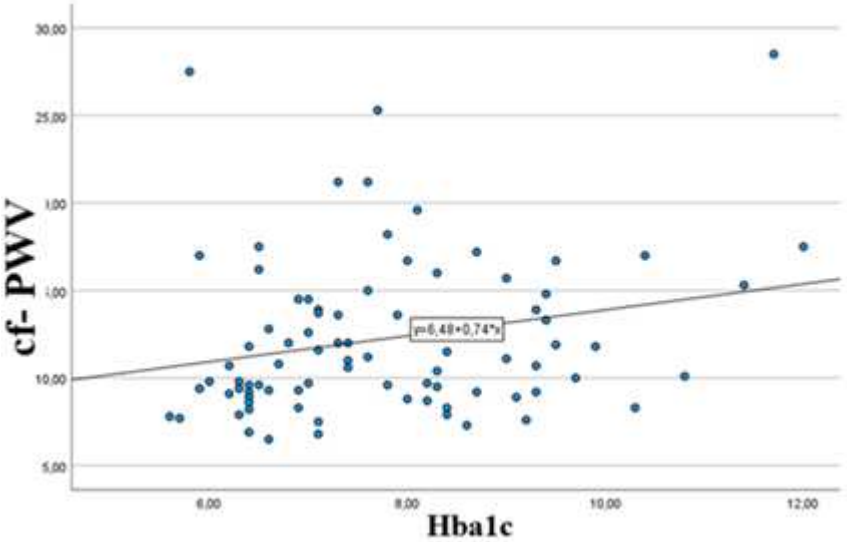


Figure 4: Correlation between diabetes duration and cf-PWV

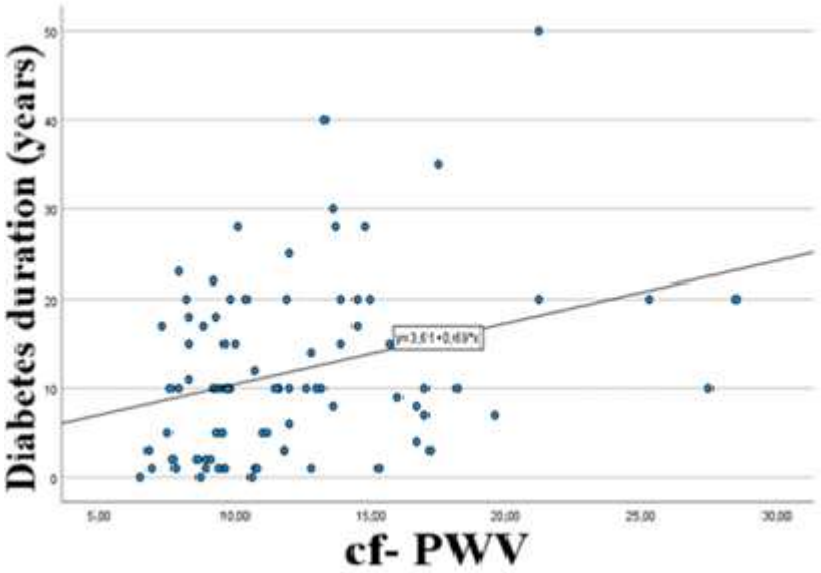


Table 1: Baseline characteristics of the study population according to HbA1c groups

Variables	HbA1c < 7.0% (n=29)	HbA1c ≥ 7.0% (n=57)	p value
Age (years), [IQR]	65,0 [60,5-69,0]	65,0 [59,0-70,0]	0,574
Male, n(%)	19 (65,7)	45 (78,9)	0,177
BMI (kg/m2), [IQR]	26,8 [23,9-31,7]	28,7 [26,3-31,0]	0,196
Diabetes duration (years), ± SD	8,2±6,9	13,9±10,3	0,012
Ejection fraction (%), [IQR]	28,0 (21,5-41,5)	30,0 (22,2-35,7)	0,671
LA diameter (mm)	43,7±5,6	42,3±5,3	0,260
LA volume (ml)	75 [55-93]	62 [49-82,5]	0,097
LAVİ, ml/m 2	42 [29-53,5]	32 [25,5-41,5]	0,037
Right atrium area, cm2, [IQR]	15,1 (11,8-21,0)	13,9 (12,4-16,7)	0,334
PASP (mmHg), [IQR]	35,0 (25,0-55,0)	27,0 (22,0-45,0)	0,050
Medical History, n(%)			
HFrEF	22 (75,9)	51 (89,5)	0,117
HFmrEF	2 (6,9)	2 (3,5)	0,600
HFpEF	5 (17,2)	4 (7)	0,158
Ischemic heart disease	20 (69)	49 (86)	0,061
Hypertension	17 (58,6)	31 (54,4)	0,709
Treatment, n(%)			
ASA	19 (65,5)	35 (61,4)	0,815
ACEi/ARB/ARNI	20 (69)	41 (71,9)	0,805
Beta blocker	26 (89,7)	54 (94,7)	0,401
SGLT-2 Inhibitors	15 (51,7)	27 (47,4)	0,702
MRA	23 (79,3)	44 (77,2)	0,823
Digoxin	1 (3,4)	6 (10,5)	0,415
Ivabradin	12 (41,4)	8 (14)	0,005
Diuretics	15 (51,7)	29 (50,9)	0,941
Statin	12 (41,4)	37 (64,9)	0,037

Insulin therapy	2 (6,9)	26 (45,6)	<0,001
Sulfonylureas	0 (0)	4 (7)	0,295
Metformin	15 (51,7)	25 (43,9)	0,489
Laboratory Findings			
Fasting glucose (mg/dl), [IQR]	125,0 (106,0-150,5)	169,5 (145,5-230,5)	<0,001
HbA1c (%), [IQR]	6,4 (6,2-6,5)	8,3 (7,6-9,0)	<0,001
eGfr, ml/dk, [IQR]	69,6 (49,1-89,9)	66,7 (44,8-81,1)	0,317
Total cholesterol, (mg/dl), [IQR]	160,0 (129,5-185,2)	142,0 (122,0-168,2)	0,337
HDL cholesterol, (mg/dl), ± SD	41,5±9,8	37,1±8,4	0,033
Creatinine (mg/dl), [IQR]	1,0 (0,8-1,3)	1,1 (0,9-1,5)	0,078
Albumin (g/dl), [IQR]	4,3 (3,8-4,6)	4,5 (4,0-4,7)	0,204
Hemoglobin (g/dl), [IQR]	13,7 (10,4-15,2)	13,9 (12,7-14,6)	0,464
NT-ProBNP (pg/ml), [IQR]	1820 (250-11564)	626 (237-1844)	0,385
cf-PWV (m/s)	9,4 (8,6-11,8)	12,0 (9,7-15,0)	0,009

Abbreviations: ACEi — Angiotensin-Converting Enzyme Inhibitor; ARB — Angiotensin Receptor Blocker; ARNI — Angiotensin Receptor–Neprilysin Inhibitor; ASA — Acetylsalicylic Acid; BMI — Body Mass Index; cf-PWV — Carotid–Femoral Pulse Wave Velocity; eGFR — Estimated Glomerular Filtration Rate; HDL — High-Density Lipoprotein; HF — Heart Failure; HFrEF — Heart Failure with Reduced Ejection Fraction; HFmrEF — Heart Failure with Mildly Reduced Ejection Fraction; HFpEF — Heart Failure with Preserved Ejection Fraction; IQR — Interquartile Range; LA — Left Atrium; LAVI — Left Atrial Volume Index; MRA — Mineralocorticoid Receptor Antagonist; NT-proBNP — N-terminal Pro–B-type Natriuretic Peptide; PASP — Pulmonary Artery Systolic Pressure; SGLT2 — Sodium–Glucose Cotransporter-2.

Table 2: Device-detected arrhythmias in HbA1c <7.0% and HbA1c ≥7.0% groups

Variables	HbA1c < 7.0% (n=29)	HbA1c ≥ 7.0% (n=57)	p value
Subclinical atrial fibrillation	8 (%27,6)	5 (%8,8)	0,029
Ventricular tachycardia	4 (%13,8)	8 (%14)	0,8
Anti-tachycardia pacing/ICD shock	1 (%3,4)	3 (%5,3)	1

Abbreviations: HbA1c — Glycated Hemoglobin; ICD — Implantable Cardioverter-Defibrillator; SCAF — Subclinical Atrial Fibrillation; VT — Ventricular Tachycardia; ATP — Anti-Tachycardia Pacing.

Table 3: Association Between Subclinical Atrial Fibrillation Detected at 6-Month Follow-Up and Baseline HbA1c Levels and Aortic Stiffness

Variables	Subclinical atrial fibrillation(+) (n=10)	Subclinical atrial fibrillation(-) (n=65)	p value
HbA1c (%), ± SD	6,9±0,7	7,9±1,5	0,004
Glucose, mg/dL, [IQR]	131 (94-160)	161 (132-217)	0,031
cf-PWV (m/s)	12,7±7,2	12,1±4,5	0,789

Abbreviations: HbA1c — Glycated Hemoglobin; cf-PWV — Carotid–Femoral Pulse Wave Velocity; IQR — Interquartile Range; SD — Standard Deviation.

Table4: Univariate and multivariate regression analyses for determinants of ΔPWV in the HbA1c ≥7.0% subgroup

	Univariate analysis		Multivariate analysis	
Baseline characteristics	β (95%CI)	P Value	β (95%CI)	P Value
Subclinical atrial fibrillation	5,85 (1,04–10,66)	0,017	6,21 (1,61–10,81)	0,009
LA volume	0,08 (0,03–0,13)	0,0035	0,06 (0,008–0,105)	0,024
Diabetes duration	-0,12 (–0,25–0,01)	0,079	–0,10 (–0,22–0,02)	0,107
Baseline cf-PWV	–0.26 (–0.58-0.05)	0,103	–0,37 (–0,71– –0,04)	0,029
Age	–0.01 (–0.16-0.14)	0,924	+0,11 (–0,07–0,28)	0,233

Abbreviations: ΔPWV — Change in Pulse Wave Velocity; HbA1c — Glycated Hemoglobin; LA — Left Atrium; cf-PWV — Carotid–Femoral Pulse Wave Velocity; CI — Confidence Interval.